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EXPERIENCE & REASON

Short communications of factual material are published here. Comments and criticisms appear as Commentaries or Letters to the Editor.

"In Medicine one must pay attention not to plausible theorizing but to experience and reason together. . . . I agree that theorizing is to be approved, provided that it is based on facts, and systematically makes its deductions from what is observed. . . . But conclusions drawn from unaided reason can hardly be serviceable; only those drawn from observed fact."

—Hippocrates, *Precepts*

Acute Brain Herniation From Lead Toxicity

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ABSTRACT

A 4-year-old black boy was admitted to the hospital with vomiting, low-grade fever, and dehydration that were thought to be caused by viral gastroenteritis. He proceeded over the next 12 hours to rapidly deteriorate with brain herniation leading to brain death. The ultimate cause of death was found to be acute lead intoxication from a swallowed foreign body.

Lead poisoning is a common cause of neurologic morbidity, with as many as 20% of children affected in some communities in the United States.[1] Children of lower socioeconomic status in inner cities and some rural communities seem to be at highest risk.[2] Most pediatric cases are the result of exposure to lead paint, although other sources such as foreign-body ingestions have become more common recently. Symptoms of lead intoxication can vary depending on the time course and severity of the ingestion as well as the age of the patient. Children with significant acute lead exposure can present with irritability, inactivity, gastrointestinal symptoms, headache, lethargy, and seizures. In rare cases, acute lead intoxication can lead to symptoms that mimic a central nervous system mass lesion.[3,4]

CASE REPORT

A 4-year-old black boy initially presented to the emergency department (ED) with a 2- to 3-day history of vomiting, as well as decreased energy, decreased oral intake, and some fever at home. No diarrhea or ill contacts were reported. His past history was significant for a history of developmental delay with microcephaly, slow weight gain, and an elevated blood lead level of 12 μg/dL at 2½ years of age. The latter resolved without treatment, and at his 4-year well-child visit (5 weeks before this admission), his blood lead level was normal at <1 μg/dL.

Initial evaluation in the ED showed him to be afebrile, with moist mucous membranes and tears and a soft abdomen. No laboratory or radiologic evaluation was performed. He was given ondansetron and a trial of oral fluids, which he tolerated. He was discharged from the hospital with a diagnosis of vomiting.

The patient returned to the ED 2 days later with continued vomiting (nonbilious) and increased sleepiness. The examination was significant for an oral temperature of 38.0°C, pulse of 132 beats per minute, respiratory rate of 28 breaths per minute, and blood pressure of 107/56 mm Hg; his weight was down 0.7 kg from 2 days before. He was noted to be lethargic but able to answer questions appropriately. His mouth was very dry, and his abdomen had decreased bowel sounds but was otherwise benign. It was also noted that he had very decreased subcutaneous tissue. His electrolyte levels were within normal limits, but his serum urea nitrogen was elevated to 35 mg/dL and his creatinine level was 0.7 mg/dL. He was given 2 fluid boluses of normal saline, after which he became more talkative. He was admitted

Key Words: lead levels, brain perfusion

Abbreviations: ED, emergency department; CT, computed tomograph

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shortly after midnight to the general pediatrics service for additional care.

The patient was seen by the attending pediatrician around the time he was sent to the inpatient unit from the ED. Examination showed him to be quiet but able to converse. He was noted to be microcephalic with somewhat low-set ears. His abdomen was soft, with slightly increased bowel sounds but no rebound tenderness or organomegaly. Capillary refill was 3 seconds, and skin turgor was noted to be fair. Intravenous fluids were continued because, with persistent vomiting, he was not tolerating oral fluids.

The following morning the patient was seen by a medical student at 6:20 AM, at which time he was still complaining of abdominal pain but had not had any further emesis. His temperature had increased to 39.1°C overnight. He was seen 3 hours later by a resident, who agreed with the benign examination and the plan to continue intravenous fluids. Abdominal and chest radiographs as well as tests for amylase and lipase levels were also ordered to further evaluate the ongoing complaints of abdominal pain. During this time he was noted to be awake for a time and told the nurse at 7:45 AM that he was "feeling better" before falling back asleep. Approximately 2½ hours later, he awoke screaming, became agitated and combative, and bit his mother. The attending physician came to evaluate the patient, at which time he was sleeping and was also noted to be congested and snoring (which were also noted at the admission examination).

Repeat morning laboratory tests showed electrolyte, serum urea nitrogen, and creatinine levels to be within normal limits. Amylase and lipase levels were also within normal limits. His hemoglobin level was 8.8 g/dL (down from 10.4 two months before at his well-child care visit), with a white blood cell count of 13 900/μL with 69 polymorphonuclear cells, 10 lymphocytes, and 20 mononuclear cells and a normal platelet estimate. Mean corpuscular volume was 82 fL.

Chest and abdominal radiographs had already been ordered that morning (but not yet obtained), and because of the new agitation, a head computed tomograph (CT) was also ordered to check for an intracranial process as a cause for the vomiting and agitation. Before the patient could be taken down for his CT, his level of consciousness decreased significantly, and he became stiff, with eye deviation to the left. He was noted to be unresponsive to stimulation. His pupils were 2 to 3 mm and reactive. Intermittent decerebrate posturing was noted. A code blue was called, and the patient's trachea was intubated. He was stabilized and transported emergently for his CT, on which mildly dilated ventricles with absence of cisterns were noted, suggesting herniation and mild obstructive hydrocephalus. No cerebral edema was identified.

The patient was initially taken to the PICU for stabilization. After consultation with our neurosurgery department, a decision was made to take the patient to surgery, but before being transferred, the patient's pupils were noted to be fixed and dilated. He received mannitol and 3% saline and was taken to surgery, where a ventriculostomy was placed. Postoperatively, his pupils remained fixed and dilated.

As mentioned above, an abdominal radiograph had been ordered before his arrest, but it was not performed. On the initial postintubation radiograph (Fig 1), an opaque body, thought to be either an electrocardiogram lead or foreign body beneath the patient in his bed, was noted in the left upper quadrant of the abdomen.

However, on subsequent radiographs, this object persisted, including on lateral views, and was eventually identified as being a foreign body, presumably within the stomach. It had a heart-shaped appearance. A heavy-metal screen had been sent off and came back 2 days later revealing a blood lead level of 180 μg/dL (reference level: <10 μg/dL).

Over the following hours, the patient had ongoing problems with hypotension requiring pharmacologic support. By that evening, on the basis of lack of respiratory effort on continuous positive airway pressure alone and a nuclear medicine perfusion scan showing no evidence of intracranial blood flow, a diagnosis of brain death was made. The patient continued to be supported over the next 3 days while the parents struggled with the reality of their child's death. Support was withdrawn 72 hours later.

The parents agreed to an autopsy, and the case was referred to the medical examiner's office. Autopsy results found the foreign body to be a piece of a charm bracelet (Fig 2), and the composition was found to be 99% lead. Cause of death was acute lead intoxication.

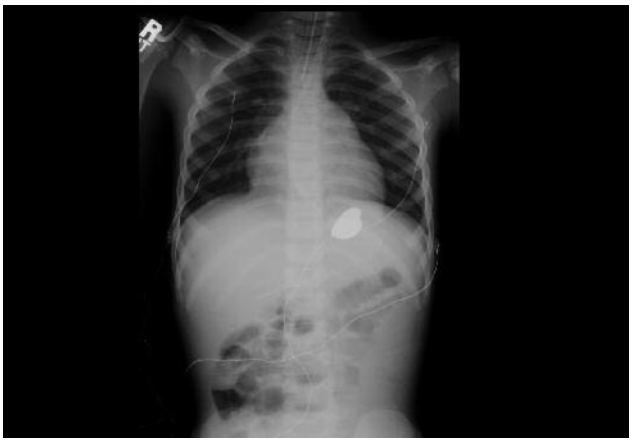

FIGURE 1
Initial chest and abdominal radiograph showing a heart-shaped object in an area of the stomach.

FIGURE 2
Replica of the heart-shaped charm found in our patient's stomach.

DISCUSSION

Lead intoxication in children continues to present a significant public health problem despite efforts to curb exposure. Up to 21% of inner-city children have lead levels above the limit of 10 μg/dL set by the Centers for Disease Control and Prevention.[2] Compared with adults, children are also at increased risk of lead toxicity. Children show proportionally great absorption of lead in the gastrointestinal tract.[5] Lead penetration of brain tissues is greater in children, and developing neuronal tissue has also been shown to be more sensitive to the toxic effects of lead.[6] Furthermore, the toxicity of lead in children manifests itself for extended periods of time despite therapy and cessation of exposure.[7,8]

Most commonly, lead intoxication in young children is attributed to ingestion of lead-based paint. However, recent reports indicate that exposure to other items such as traditional medications, toys, and trinkets can result in lead intoxication.[9] In 2004, a 4-year-old boy presented with symptoms of lead intoxication and a blood lead level of 123 μg/dL.[10] A medallion obtained from a toy vending machine was retrieved from the boy's stomach. In our case, the child ingested a heart-shaped locket that contained 99% lead. Interestingly, similar lockets tested subsequently showed an alarming variety in lead content, possibly because of varied manufacturing.

The clinical presentation of lead intoxication can vary dramatically. Children with mild, chronic lead poisoning (ie, serum levels >10 μg/dL) may show few clinical symptoms yet still suffer neurocognitive impairments that are permanent and irreversible. However, children with more significant acute lead intoxication can present with more severe symptoms such as irritability, lethargy, vomiting, seizures, and coma.[6] Cerebral edema is a common finding in children with lead encephalopathy, and adult cases have also shown cerebral calcifications.[11] A recent review cited several studies showing that although lead toxicity is complex, several mechanisms such as enhanced apoptosis, disruption of the blood-brain barrier, and oxidative stress can lead to cerebral edema.[12] Often, cerebral edema found in lead intoxication is acute and transient. Sharma et al[13] described 2 infants who presented with neurologic signs and bulging fontanelles. One child's CT showed dilated ventricles and a swollen cerebellar vermis acting as a mass lesion. Both children showed radiologic evidence of lead intoxication (lead lines in bones) and were treated with chelation therapy as well as other supportive measures.

Less commonly, children may present with more severe symptoms. Mirando and Ranasinghe[14] described the case of an 11-month-old patient with acute lead intoxication leading to cerebral herniation and death. Interestingly, there have also been cases of lead intoxication mimicking the signs and symptoms of central nervous system mass lesions. Powers et al[3] described a 47-year-old patient with acute and chronic lead intoxication who presented with focal neurologic signs and decreased mental status. A CT of the brain showed midline shift and suggested a mass lesion of the left temporoparietal area. At the time of surgical intervention, no obvious lesion was discovered, and the patient died 2 days later. Autopsy findings were consistent with edema of the cerebral hemispheres but without an obvious mass. Pappas et al[4] reported a 9-month-old who presented with irritability, lethargy, and a bulging fontanelle. A CT of the brain revealed massive ventriculomegaly and a compressed fourth ventricle as well as low attenuation of the cerebellar white matter. The patient's cerebral edema progressed, and he died 24 hours later. Laboratory studies revealed elevated lead levels thought to be a result of ingestion of several "tonics" that were sent to the family from India.

The patient described in this case study presented with subtle signs of lead intoxication including vomiting and lethargy. He also had a history of fever, thus complicating the diagnosis. One unique aspect of our patient's course is that his mental status changes occurred over such a short period of time, thus precluding chelation therapy. Brain imaging in our patient demonstrated signs of obstructive hydrocephalus with mild dilatation of the ventricles. Also, very little cerebral edema was seen on his head CT, inconsistent with typical radiologic findings.

This case, in conjunction with the literature, should increase our awareness of lead intoxication as a possible cause of increased intracranial pressure. We believe that in patients with vomiting and neurologic changes, lead intoxication should be considered as a diagnosis even when brain imaging either shows no cerebral edema or shows signs of a mass lesion. Providers should also consider lead toxicity in patients with unexplained symptoms such as vomiting, developmental delay, hearing loss, behavioral problems, seizures, or anemia. In addition, it shows that adequate public health surveillance is crucial if we are to reduce the availability of lead-based objects that children find so enticing.

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